

At page 6, line 7, please insert the following:

-- BRIEF DESCRIPTION OF THE DRAWINGS

Figure 1 shows the results of biological experiments in which oedema-suppression activity of hydrocortisone in a commercial reference creme (open symbols) was tested against the same amount of identical drug in highly deformable lipid vesicles (Transfersomes) (closed symbols) in mice. The upper panel contains time-dependence ("pharmacodynamic") data, whereas the lower panel gives the dose dependency measured 16 h after the drug application. Data points give the mean values for 3-4 animals.

Figure 2 illustrates the suppression of the arachidonic acid-induced oedema by dexamethasone in a commercial creme (open symbols) or in Transfersomes (closed symbols) as a function of the time after drug administration (upper panel) or of the epicutaneously applied drug dose (lower panel).

Figure 3 provides information related to that given in figures 1 and 2, but pertaining to a different glucocorticosteroide, triamcinolone acetonide.

Figure 4 presents the results of dose and time dependence measurements for triamcinolone-acetonide applied in a commercial creme (open symbols) or in Transfersomes (closed symbols) on one forearm of a healthy human volunteer. The read-out was the extent of skin blanching caused by the drug, at the tested doses as given in insets.

Figure 5 shows dexamethasone penetration profile in murine skin in vivo (left panel) or in a pig skin ex vivo (right panel). Open symbols were measured with a commercial creme and closed symbols with the suspension of dexamethasone-loaded Transfersomes.

Figure 6 demonstrates the level of corticosteroid accumulation (retention) in the skin after different drugs' application on the organ surface by means of Transfersomes (closed down-arrow: the stratum corneum; closed up-arrow: the skin stripped free of the stratum corneum; open diamond: total drug amount in the entire skin (= the sum of the former two)).

Figure 7 illustrates (pharmaco)kinetics of transcutaneous transport of various corticosteroids, as assessed by measuring the drug derived radioactivity in the serum, following the topical drug administration with ultradeformable vesicles on (closed symbols) or under (open symbols) intact murine skin. Data points give the mean values for 3-4 animals and vertical bars give standard error of the mean. The applied drug dose in relative units is given in insets.

Figure 8 provides some representative data on biological, anti-oedema activity of triamcinolone-acetonide applied on the skin in a commercial lotion or in conventional lipid vesicles, liposomes (lower panel) or else in highly deformable mixed lipid vesicles, Transfersomes (upper panel). All results were determined in the arachidonic acid induced murine ear oedema model. The topically applied doses are given in the panels. Formulation B was based on oleic acid rather than on phospholipids, as the main carrier ingredient.

Figure 9 shows the results of skin atrophy measurements in healthy human volunteers, treated for 6 weeks, twice daily, epicutaneously with two triamcinolone acetonide formulations (TAC I and TAC II), differing in antioxidant composition, as specified in Examples 53 - 56, "pre" relating to the pre-treatment phase, "rec" to the recovery phase, and "x" to the number of weeks of recovery.

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IN THE CLAIMS:

Please cancel claims 26-34, 36-38 and 47-50, without prejudice.

Please amend claims 1, 5, 7, 9, 11 and 39 to read as follows:

1. A formulation comprising penetrants being capable of penetrating the pores of a barrier, even when the average diameter of said pores is smaller than the average diameter of said penetrants, wherein said penetrants can transport agents or enable agent penetration through said pores after said penetrants have entered said pores, wherein the formulation further comprises